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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

CONTENTS:

THE PSYCHONEUROTIC VETERAN 165
Major Sidney Rosenbliett, M.C., *and*
Captain Joseph E. Pisetsky, M.C., Roseburg, Oregon

NEUROLOGICAL COMPLICATIONS OF ESSENTIAL PURPURA HAEMORRHAGICA 170
Lt. Joseph D. Teicher, M.C. (S) USNR

THE PHYSICIAN'S FIELD WORKERS FOR MENTAL HEALTH 175
Robert L. Sutherland, Ph.D., Austin, Texas

NEUROSIS—A PROGRESSIVE OR SELF-LIMITING DISEASE? 177
Edmund Bergler, M.D., New York, New York

NEUROLOGICAL CLINICAL PATHOLOGICAL CONFERENCE 181

SANITARIA, NEWSNOTES, BOOK REVIEW

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

The Psychoneurotic Veteran

MAJOR SIDNEY ROSENBLIETT, M.C.* *and* CAPTAIN JOSEPH E. PISETSKY, M.C.*
Roseburg, Oregon

The accelerated rate of discharge of service men and the rapid demobilization of the armed forces has posed the problem of treating the increasing number of veterans who are designated as psychoneurotic. Already there are over two hundred thousand and the number bids fair to rise much higher. From a therapeutic viewpoint, this involves a staggering amount of reconstructive mental engineering before these ailing veterans can be reintegrated into the body of our population without disastrous results. At the time of induction into the armed forces the individual had to be reoriented and adjusted to the new milieu. His morale had to be strengthened so that he could work for a common purpose. A similar situation confronts us now during this vast demobilization and again a realistic and positive approach must be adopted to weld the individual into the framework of our society without creating a loose or faulty joint.

Every dischargee will be confronted with the necessity for psychological armoring of varying degrees. Many may have been exposed to the monotony of inactivity and isolation, while others have experienced the excessive fatigue and anxiety laden atmosphere of combat. However, the nearness to home, release from military discipline and the return to former ways of life will be a powerful desensitizing agent for most veterans. In the initial stages of his transition from service man to civilian, several agencies such as the Red Cross, U. S. Employment Office, the Vocational and Rehabilitation Offices (both Federal and State) play an important part in helping the psychoneurotic veteran make a satisfactory social adjustment. Case discussion, advice, and manipulation of the patient's environment through recognized channels may be sufficient to establish the veteran as student, apprentice, industrial worker, or as a small enterpreneur. This elementary program must be implemented by a capable local and national leadership which comprehends in a positive dynamic way the needs of the veteran who clamors for security and recognition.

Many authorities assert that the situation is exaggerated, while others believe that insufficient preparation has been made to care for these individuals. Obviously the extent of the problem will depend on the economic situation. However, even under the best economic circumstances it will be huge. Ginsburg[1] has indicated in a preliminary report the magnitude of the need for treatment in the New York area. Utilizing a random sample of 309 discharged men, he found that 235 needed treatment; only 25 per cent of these desired treatment, and only 16 per cent were receiving help. In contrast, of 74 per cent of dischargees with medical and surgical conditions, between 80 and 90 per cent were receiving the medical and surgical assistance they required. This marked disparity in treatment facilities is general throughout the country and presents a provocative challenge for the future. Apathy and delay in treatment will be disastrous.

In 300 cases referred to Rennie[2] for psy-

*U. S. Veterans Administration.

chiatric rehabilitation, very similar problems were encountered: "A sense of stigma because of psychiatric discharge; a need for proper employment, a lack of recreational facilities and companionship, uncertainty as to how to formulate their discharge, family overconcern or rejection, compensation procedures, a feeling of neglect and varying degrees of disability due to unresolved psychoneurotic or psychotic symptoms." Out of the group under study 119 cases, or 54 per cent had symptoms characteristic of psychoneurosis, particularly of the anxiety reaction type. There were fewer cases of the other forms: hypochondriasis, 9; hysteria, 8; obsessive-compulsive, 2; "battle neurosis," 2. The infrequency of the latter group is striking. A high percentage of those who fall into the "battle neurosis," combat fatigue, or operational fatigue category attempts to make a good social adjustment and usually are more interested in getting well than in seeking compensation. In Rennie's group only 10 per cent seemed to be interested in compensation. Of the 70 cases, who manifested various gradations of improvement, 39 per cent were seen in one consultation and 6 per cent not more than three times. Depression, anxiety, and hypochondriacal reactions, demanded more prolonged, but "brief psychotherapy". These results are heartening and give an indication as to the scope of future therapeutic possibilities.

The recent ardent appeal by Smith and Wood[3] to foster a greater participation of the general practitioner in the treatment of psychoneurotic veterans is most laudable. There are many veterans, as indicated by Rennie, who need only some bolstering advice and then can be steered to the proper agency without further treatment. For this large group of mild psychoneurotics the general practitioner can do yeoman work. However, in tackling the more involved problems in dynamics, homely advice and the reading of specific texts is not sufficient. The family Doctor can be extremely helpful in reorienting the uninitiated but it must always be borne in mind that inadequate or faltering therapy may lead the veteran into a chronic state. The crux of the problem lies in properly sizing up and evaluating the condition. The general practitioner can increase his finesse in segregating those cases which need skilled treatment. There is room for much education along these lines. A bugaboo for the practitioner is the apprehension of long sessions with the psychoneurotic. The physician can estimate the time he can devote to his organically ill patients, (and in these busy times he must be chary of his hours), but when he contemplates delving into a psychiatric history he has to be fortified with a faith that his restricted efforts will be of proportionate value.

Case II following, is a prototype of the veteran appearing for pension examination, who has valiantly knocked at the door of many practitioners, and has spent large sums of money frequently earned under the stress of most harassing symptoms only to discover that the symptoms continue unabated. It is realized that the physician's attempts may be fruitless and sterile and Rennie's statistics argue for caution. Yet even he would agree that the practitioner is an important link in the complex chain of therapeutic agencies. Since optimism is the keynote, every physician must be coaxed into this gigantic work. The experiment is worth the trial.

In this connection, four cases are presented outlining the general problems confronted in a large group of psychoneurotics. The authors have seen and examined hundreds of these cases at least half of whom are amenable to treatment.

Case I: E. K., a 24 year old veteran, was examined at U. S. V. A. F., Sheridan, Wyoming, on December 14, 1944. On June 4, 1943, while on furlough from the Army, veteran entered a burning building to rescue someone. He sustained burns of the head, ears, face, neck, hands, wrists, groins, hips, buttocks, genitalia, and both lower extremities down to the ankles. He was treated and 450 square inches of skin had to be grafted.

He lay in the hospital about one year until these burns healed completely and then, due to scarring, a paralysis of the left foot resulted so that he was required to use a brace. He also had deformities of his face, back and abdomen. Previous to service he had been an excellent basket ball player. Now he was crippled and unable to pursue any sports or employment and he brooded over his incapacitation, losing interest in outside activities and in those about him and lacking in ambition. He was preoccupied with his body. His face had taken on a cadaverous expression and even his friends stated he "looked more dead than alive." Emotionally, veteran was dull and apa-

thetic with little inclination to smile. He was in good contact but recited the story of his misfortune in unanimated fashion. He was disturbed by a severe insomnia. He also displayed a fairly marked axillary hyperhidrosis and some vasomotor instability.

During veteran's long period of hospitalization his mental condition had not been recognized and now five months after discharge he was exhibiting a pre-psychotic picture with no provision made for his treatment.

Case II: F. R. L., a 29 year old veteran was examined at U. S. V. A. F., Sheridan, Wyoming, on October 26, 1944, 14 months after his discharge from the Navy. He was receiving a pension which he utilized for the most part in obtaining treatment from a private physician with little relief. He had had innumerable jobs since discharge, earning up to $50 weekly, but he was unable to remain long on one job. Patient, being the youngest child in his family, had received much preferential treatment. There was a good deal of turmoil at home and he drank heavily; he still bites his fingernails. In August 1942, at Guadalcanal, after a naval engagement he was helping to rescue survivors from the sea. While pulling a survivor out of the water a shark snatched the man out of his grasp and pulled him under. Veteran soon thereafter became fearful, couldn't sleep and had frightful dreams, dealing mostly with sharks and Japs, and he would hear the victims crying out about the sharks. Since discharge he has had difficulty in holding a job for more than a few months; he was tremulous and suffered from mid-occipital headaches; he had "convulsions" during which he grinds his teeth, tears up pillows, pulls everything apart, thrashes about in bed, and is fearful of harming someone during an attack. He tires easily, suffers from palpitation and dyspnoea. Objectively he reveals a good deal of anxiety, apprehension, restlessness, emotional instability, manual tremulousness, tachycardia, and a preoccupation with his symptoms. Veteran is eager to get well so that he can work and get along with people.

Case III: R. E. R., a 25 year old white veteran, was admitted to the U. S. V. A., Roseburg, Oregon, August 22, 1945, six weeks after his discharge from service. An important fact in his family history is that his father suffered from heart trouble. His past personal history shows that he had always been well adjusted, and had none of the early neuropathic traits. He served in the Air Corps, completing 30 missions as a pilot. He had had two flying accidents. The first while in training when he had to make a forced landing in the desert. He was successful in getting the plane down and was not hurt. The incident is significant however, because he noticed a peculiar sensation in his heart causing him to think that he had heart trouble. The second accident occurred in combat when his engine "conked out" and he made a forced landing, damaging the plane but without injuring anyone.

The patient's first "heart attack" occurred in April 1945, while on a flight. Nothing unusual happened which could account for this attack. He was finally discharged from service July 1, 1945, at which time he said nothing about his cardiac difficulties. On August 15, 1945 he had a very severe attack during which he experienced palpitation, was short of breath and had the sensation that his arms and legs were going to sleep. His doctor told him he had heart trouble explaining that his heart had become accustomed to large quantities of oxygen because of his flying experiences. This information aggravated his condition and he demanded an oxygen tank while being transported to the hospital. Investigation in the hospital revealed no organic cardiac signs. The difficulties in back of his heart trouble were his marital problems. He was married in March 1943, and lived with his wife for short periods—no more than three weeks at a time. When he returned to this country in October 1944, she asked for a divorce. Since then he had been going out with a girl he had known earlier, when he was about 21. At that time this girl wanted to marry him but he did not concur. Presently this girl is married and her husband is over seas. He feels this girl still loves him and that he loves her. Now he is desperate since this girl does not wish to leave her husband and will not consider divorce because she is Catholic. When these facts were brought out the patient was under great tension. While discussing his marital problem he gained some superficial insight. However, he disliked confinement in a mental institution and demanded his discharge, which was complied with three days after admission.

It is unfortunate that the patient was unwilling to remain in the hospital longer, so that the conflicts which were uncovered could be fully discussed with him. He was willing to return to the hospital for further treatment on an outpatient basis but this was not possible under existing arrangements.

Case IV: P. R. S., a 26 year old veteran, was admitted to the hospital on May 25, 1945, after waking from a fugue during which he traveled from New York City to the Pacific Coast. He was picked up by police and was hospitalized for a period of four days during which he was in a trance and neither ate nor spoke. A review of his family history shows that there was considerable conflict between the patient's paternal grandmother and his mother. The latter was Catholic, and his father's family were Protestant. He often heard his mother and grandmother speak disparagingly of each other. The grandmother frequently criticised his parents' marriage and spoke against marriage in general. His mother was not inclined to worry but she often displayed her "Irish hot temper." He describes his brother

as of an entirely different temperament; that the brother was easy going, friendly, a good mixer with the opposite sex, and inclined to be very thrifty. Patient denied any early neurotic traits such as nocturnal enuresis, sleep walking, sleep talking, or nightmares. At the age of 15 he had reached the fifth grade when he quit school to go to work. He later entered night school and continued his education. He avoided girls and gave as his reason that he had to take care of his younger brother. He denied all heterosexual experiences.

Patient had an amnestic episode in 1943, following the crash of an airplane which he had failed to inspect. This gave rise to guilt feelings and he became dazed and confused, and was ultimately discharged from service. Shortly after returning home his mother died of a throat cancer. Following her death he went to New York but while working there, he began to feel anxious and felt the need to visit his sisters and father. The next thing he knew he found himself in the Veterans' Hospital at Portland, Oregon. He was told that he had been in a stupor for four days and that he had not eaten. There were scratches on his wrists which were believed to have been the result of a suicide attempt, about which, however, patient denied all knowledge. He was eventually admitted to the U. S. Veterans Administration, Roseburg, Oregon, by transfer. On admission he had difficulty in swallowing, expressing an aversion for food, particularly milk. This, however, disappeared in the course of 24 hours. He then seemed elated and cheerful, but at the same time was very anxious to leave the hospital.

Through hypnosis all the events of the first fugue following the airplane accident were brought out in detail. Equal success was obtained in eliciting the details of his second fugue. In addition, an early occurrence was uncovered in which patient once said to his mother: "'I hope when you die, you die of a cancer.' I killed my mother just as if I drove a knife through her, just as true! Just as true! Just as true! I hope she has forgiven me."

In this patient considerable success was achieved by means of hypnosis disclosing the events that transpired during both amnesic episodes. Much of the psychodynamics was also revealed. Unfortunately, patient did not like the environment of a mental institution and demanded his release and was finally discharged June 20, 1945, four weeks after admission.

This patient also was eager to return to the hospital for treatment on an out-patient basis and no doubt would have derived great benefit from further explanation and elucidation of the many conflicts that were at the bottom of his condition.

Of the four cases, the first two were examined for pension purposes; the others were hospitalized in a psychiatric institution. All four were in need of treatment which could be rendered on an out-patient basis. The last two illustrate that institutions are not particularly suitable for care and treatment of the psychoneurotic veteran.

These cases demonstrate vividly that improvement is not spontaneous and is not contingent on the veteran's return home. The Army Times of September 29, 1945, reporting a survey of 500 Veterans made by W. Menninger, Brill, and Tate arrive at the same conclusion. These authors found that "only 11.4 per cent of the emotionally unstable veterans showed improvement after discharge while 63.6 per cent reported no change and 24.7 per cent said their condition had become worse." The study also indicates the tendency for chronicity to set in rapidly.

There is as yet no clearly formulated plan as to the type of therapy indicated for each category of psychoneurotic disability. Psychotherapy is a term used to cover all such methods of treatment. The aim of therapy is to correct the character structure so as to make possible a satisfactory social adjustment. Unfortunately, the treatment of psychoneuroses and functional disorders is as yet in its infancy, and few physicians have been properly trained to cope with them. Our medical schools are partly responsible for this. In general medical practice at least 40 per cent of the patients encountered have difficulties of a functional nature, yet less than 5 per cent of the time spent in medical schools is devoted to their study. Present medical thinking still adheres to the organic tradition. Physicians, as a general rule, will search and search before being willing to concede the absence of physical disease when dealing with an obscure illness. Military experience has altered this outlook somewhat. Many army hospitals now require a neuropsychiatric evaluation of all patients who are to undergo elective surgery and the frequency of neuropsychiatric consultations in medical cases can be attested to by any psychiatrist working in a military hospital. To understand the dynamics of the psychic conflicts which underly the psychoneurosis and the functional disabilities, requires long and intensive study and is time consuming. For this reason any program that undertakes the treatment

of the psychoneurotic veteran must provide for adequate and intensive training of physicians interested in this field.

Besides the pre- and post-treatment evaluation of a neurotic condition, other criteria, such as the goals of treatment have to be considered. In view of the multitudes requiring treatment, short term forms of therapy will have to be applied. Treatment must be pursued along psychodynamic lines and therefore certain techniques, such as hypnosis, hypno-analysis and narcosynthesis are the methods of choice. The psychiatrist must work out an approach adapted to the particular case and the time at his disposal.

Interesting also, is Alexander's[4] advice to use supportive therapy where a constitutional or a weak ego exists. He believes that inferiority feelings can be strengthened by reassurance, anxiety mitigated by assuming a protective role, and guilt feeling relieved by permissive attitudes. Of fundamental importance also, is his recognition of the fact that any individual may develop a neurosis under sufficiently overwhelming stress or traumatic situations, even though in the past, neuropathic backgrounds did not exist. For many years Schilder[5] advocated the use of short psychotherapy lasting up to 30 days and he frequently combined this technique with hypnosis. It has gradually become more obvious that the "inner repetitive core" (Kubie)[6] does not have to be reached to achieve a good measure of therapeutic success. Deutsch[7] has proposed a worthy goal in the treatment of traumatic neuroses, which is "to bring back the patient to the condition in which he was before he had the traumatic experience."

A distressing obstacle that the psychotherapist encounters is the estimation of his results. Many investigators have been thwarted for a long time in their efforts to measure objectively their therapeutic achievements. A completely adequate solution may be remote, but certain facets of accomplishment can now be measured. A very helpful instrument is the Rorschach test which has been adopted by Ross.[8] Its extended utilization in the future in comparing pre- and post-treatment states relative to improvement will determine the value of various methods. The recent application by Hoch[9] of psychogalvanic registration in traumatic neuroses is a refreshing approach to the difficult problem of visualizing objectively, apprehension, tension and its vegetative manifestations. Hoch was able to show the influence and value of narco-suggestion and narco-analysis in certain types of cases. He has also clearly demonstrated the existence of marked apprehension in patients suffering from gastric ulcer. This method should disclose many other facts concerning other psychosomatic disorders which now elude us.

The adoption of "scatter analysis" as elaborated by Rappaport[10] and Wechsler[11] in their psychological testing would be an excellent adjunct to other methods. Without a satisfactory objective method for determining improvement in a patient's condition, future progress in the treatment of functional disorders will be seriously hampered. On the one hand, there is the group of patients who assert that they have improved and recovered because they have no sincere desire to continue treatment. On the other hand, there are those who state that they do not feel better where all objective signs point to the contrary. In addition to the methods already mentioned the social service workers can render valuable service by obtaining further objective evidence of improvement by reports from patient's family and friends.

A cursory survey of the problem of psychoneuroses in veterans will reveal that the acute panic, anxiety and the exhaustion pictures, seen at the front or at the rear echelon, will have cleared up to some extent and the problem of situational adjustment will have been considerably mitigated by the return home. However, Murray[12] points out that the traumatic experiences of military life will in many, be the basis of later neuroses just as the traumatic emotional experiences of early childhood lead to adult neuroses. A basic policy therefore must be adopted to forestall some of the failures subsequent to World War I. At that time the guidance was inadequate and the vocational training recommended frequently was not planned to exclude the inadaptables. To prevent this impasse from recurring, and as a prophylactic against the chronicity of the psychoneuroses, a well organized mental hygiene program of extensive proportions must be initiated. An important instrument in this program must be the Out-

Patient Mental Hygiene Clinic. However, an absolute prerequisite is a corps of well trained psychiatrists. This is necessary to prevent the incrustation of a mild neurosis with the barnacles of an iatrogenic neurotic superimposition.

Hamill[13] who has had more than a generation of experience in the examination of veterans, artfully recommends that "if these psychoneurotic conditions were not so recognized they would be less common." This admonition not only exposes the pitfalls in the handling of the psychoneurotic veteran but also indicates how to approach this type of veteran, who mistakenly seeks a "crutch" for his nervousness rather than rational treatment with its greater regard in personal happiness and increased earning capacity.

SUMMARY

1. Large numbers of psychoneurotic veterans will need guidance and treatment.

2. Private practitioners can contribute greatly by recognizing the psychoneuroses and referring them to the proper agencies.

3. Established agencies as Red Cross, Vocational, Rehabilitation, etc., will advise on primary levels.

4. Mental Hygiene Clinics manned by highly trained men versed in psychodynamics should conduct the therapeutic programs.

5. Four typical cases out of hundreds of similar ones were presented as prototypes of cases which could be handled on an outpatient basis.

6. The goals and objective evaluation of treatment must be better defined.

7. Narcosynthesis, hypnosis, group and brief psychotherapy will be the methods of choice.

Authors' Note: This article is published with permission of the Acting Surgeon General, Veterans Administration, who, however, assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

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Neurological Complications of Essential Purpura Haemorrhagica

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There is much in medical literature on the complications of purpura haemorrhagica. Bleeding into the viscera, skin, lungs and uterus are listed as of not infrequent occurrence. Occasionally, there is mention of hemorrhage in the brain or adrenals. The eminent hematologist, Piney has stated "Bleeding into the substance of any internal organ may cause most puzzling symptoms, but fortunately the nervous system is rarely involved." To substantiate this statement, a review of the literature of the past fifteen years reveals a marked paucity of references to cerebral complications of purpura haemorrhagica. This paper presents two unusual cases of essential purpura haemorrhagica with intracranial complications and recovery following splenectomy and, in one case, evacuation of an intracerebral clot. There are obvious and unfortunate shortcomings in the case material; these were unavoidable but do not seriously impair the value and interest of the cases.

Before proceeding to the actual report of

the cases, it might be well to present a brief consideration of the nature of the disease.

Purpura haemorrhagica, first described by Werlhof in 1740, is variously known as Werlhof's disease or thrombocytopenic purpura. This disease may be essential or secondary to known pathological causes such as drugs, infections, toxic factors, etc. We are concerned with the essential form. The disease is most common in children but may occur at any age. In the acute form, the onset may be feverish and an attack may last from two weeks to three months. There is a chronic form where attacks may continue for years or may occur intermittently with intervals of years between attacks.

The clinical picture is characterized by purpura which may be 1-4 mm. in diameter, spontaneous ecchymoses and extravasations of blood into the mucous membranes. The striking laboratory features are the marked reduction in platelets and the prolonged bleeding time. The platelets may fall before a hemorrhage; in intermittent forms, the platelet count may be normal between attacks. In the chronic form, the platelet count may be permanently low.

In the cases to be described, the upper limit of normal coagulation time via capillary tube is four minutes. The normal bleeding time is about three minutes. The normal range of platelet count via direct smear method is 200,000 - 400,000 per cu. mm. Normal prothrombin time via Quick's method is thirty seconds. A normal clot is firm, retracts and extrudes serum. In purpura haemorrhagica the bleeding time is prolonged; the clot, while firm, does not retract or extrude serum; coagulation time is normal; the capillary resistance test is positive.

The tendency to spontaneous hemorrhages and the prolonged bleeding time are cardinal features of the disease rather than the low platelet count, which is often low even after splenectomy renders bleeding time normal and clot retraction good.

The course of the disease may be studded with remissions and exacerbations. Frequently there is a spontaneous remission. The symptoms may be mild and resolve or the attack may be fulminating and result in death. As is well known, splenectomy has been of great therapeutic value in cases of intermittent attacks, of dubious value in chronic cases and a matter of individual, delicate judgment in acute cases.

There is no pathognomonic pathological picture reflected in the body organs in essential purpura haemorrhagica. Longcope[1] pointed out that in the central nervous system hemorrhages are usually multiple and vary in size from petechiae to large extravasations.

The reduction in platelets disturbs the blood physiology and results in the abnormal hematological findings. What happens to the platelets? Why are they reduced? Most theories group about two probabilities. 1) There is abnormal activity of the mechanics responsible for platelet reduction. This is probably the reticuloendothelial system, of which the spleen is the notable member. In this connection, it is interesting to note that sometimes after splenectomy, platelets fall again but there is no prolonged bleeding and clot retraction is good. 2) There is diminished production from the megakaryocytes of the bone marrow. Perhaps the primary disorder is a defect in the capillary walls and platelets are deposited in an attempt to repair the damage, which permits only a limited number in the blood. It is safe to say the exact etiology has not been determined.

In explanation of these theories the literature has been sparse. Longcope,[1] in 1919, classified the cases into four main groups of symptoms and signs: a) those presenting chiefly the appearance of meningitis with slight focal or generalized neurologic signs, b) those exhibiting definite localizing signs among which hemiplegia is common (probably the largest group), c) those exhibiting coma, sometimes with convulsions, d) those offering no definite neurologic symptoms or signs and the hemorrhage is discovered unexpectedly at autopsy. Strauss and Tarachow[2] reviewed 105 cases of spontaneous subarachnoid hemorrhage and record only one case due to thrombocytopenic purpura with a terminal cerebral hemorrhage. Geiger[3] reported only 25 cases of intracranial hemorrhage complicating thrombocytopenic purpura up to 1932. He added two cases, one who developed a hemiplegia and died; another dying in convulsions. Garvey and Stephens[4] reported a series of 30 cases of thrombocytopenic purpura with 10 deaths, seven of which

were due to intracranial hemorrhage. They reported one case with intracranial bleeding which recovered following splenectomy. Gitt and Weiss[5] reported a case with subarachnoid hemorrhage presenting the appearance of a meningitis with generalized neurological findings. This patient recovered with no complications, only after a splenectomy.

The case presentations are as follows:

Case 1: R.H.E., Age 20 years, White male. Admitted to the sick bay on September 5 with diagnosis of septicemia with cerebral involvement.

September 5: About 3 weeks prior to admission he bruised his right arm and left leg in the gymnasium and marked ecchymoses were noted. They were slow in clearing. Two days before admission he noted small red spots on his legs, complained of sore throat, headaches, fever, chilly sensations, anorexia. Just before admission he vomited blood. Physical examination on admission showed as positive findings, pharynx and tonsillar fossa to be clotted with blood, pharynx injected, haemorrhagic purpuric spots on both legs from hips down and ecchymoses on the inner aspect of right arm and left thigh. Spleen was not enlarged. Temperature was 99.4°.

That night he had three minor convulsive seizures in five hours. The next day he developed a paralysis of the right hand, ptosis of right eyelid and thick speech. Temperature rose to 100.4°. Available laboratory data showed bleeding time: 1 minute, 10 seconds; coagulation time: 4 minutes, 5 seconds; WBC 6,500; lymphocytes 35%. He was given 100,000 units of penicillin intramuscularly and transferred to the hospital on September 6.

September 6: No additional history was obtained at the hospital. There was no family history of bleeding diseases and the patient had never before had a bleeding episode nor was he exposed to any drugs or toxic chemicals. A chest plate showed haziness in the right costophrenic angle. A blood count was: RBC 3,000,000; WBC 5,100; Hg 61%; Smear: polys 70%, lymphs 18%, eosinophils 2%, monocytes 7%, bands 3%. A scarcity of platelets was noted on the blood smear. An occasional RBC showed polychromatophilia and stippling. The impression prevailed that this was a blood dyscrasia with cerebral bleeding. He was continued with penicillin 100,000 units every two hours. Later in the day, a platelet count was 450,000/cu. mm. Other laboratory data was as follows: Urinalysis: Acid reaction; S.G. 1023; trace of albumin; sugar absent; Microscopic: Loaded with red blood cells. Prothrombin time: 57 seconds (control 31 seconds). NPN 29 mgm. %. Icteric Index 5. Blood culture: No growth. Kahn negative.

September 7: After a restless night and frequent mild convulsive seizures he was transfused with 500 cc. of whole blood. Further studies in the meantime showed a platelet count of 63,000. Reticulocyte count was 1.8%. Prothrombin time: 34½ seconds (control 27 seconds). The urine was dark colored without gross hematuria. S.G. 1023; albumin: 2 +; Sugar negative; numerous rbc/hpf on microscopic. Bleeding time was 30 minutes; clotting time was 10½ minutes. A chest plate showed thickening of the right hilar region with "pneumonitis" present. The spleen was not enlarged. The cortical symptoms were progressing. The diagnosis of purpura haemorrhagica or thrombocytopenic purpura was considered established. In view of the steady worsening of the patient's condition, splenectomy was advised. Before operation, only rare platelets could be discovered on a stained smear.

Via a left rectus incision the spleen was removed. There was considerable bleeding throughout the various stages of the operation. Patient's condition was fair after the operation, but his neurologic status did not vary from the findings on September 5. The spleen showed no specific abnormalities on pathological examination.

September 8: He was more alert and laboratory findings showed improved status: Prothrombin time: 33 seconds (control 22 seconds). Clotting time: 3 minutes, 35 seconds. Bleeding time: 6 minutes, 5 seconds. Platelet count: 62,000/cu. mm.

September 9: Further studies: Prothrombin time: 30 seconds (control 20 seconds). Clotting time: 3 minutes, 20 seconds. Bleeding time: 2 minutes, 10 seconds. Platelet count: 326,000/cu. mm. Blood culture studies were normal. His general condition improved rapidly.

September 10: Further studies on this day: (*a. m.*), Prothrombin time: 27 seconds (control 30 seconds); coagulation time: 4 minutes, 10 seconds. Bleeding time: 5 minutes, 25 seconds. Platelets: 272,000. (*p. m.*), Prothrombin time: 27 seconds (control 32 seconds). Coagulation time: 3 minutes, 15 seconds. Bleeding time: 2 minutes, 10 seconds. Platelets: 232,000.

September 11: On this date, for the first time, the neurologist was consulted. It was noted that there was a right faciobrachial paresis with increase in deep reflexes in the right arm; diminished abdominals on the right. No Babinski sign present. Sensation was intact. Visual fields, ocular movements, fundi and pupillary reactions normal. No aphasia. It was felt that there was an irritative vascular lesion in the left frontal (arm-face) cortex producing these symptoms as well as the seizures the patient sustained. The patient gradually improved. Sutures were removed on September 15. He began to use his right arm. He was transferred to the neurological service.

September 12: Prothrombin time: 28 seconds (control 28 seconds). Coagulation time: 1 minute, 50 seconds. Bleeding time: 5 minutes, 34 seconds. Platelets: 234,000/cu. mm.

September 13: Prothrombin time: 20 seconds (control 20 seconds). Coagulation time: 2 minutes, 15 seconds. Bleeding time: 1 minute, 45 seconds. Platelets: 352,000/cu. mm.

September 21: He had had no seizures since

the operation. Function in the right arm and facial paresis were improving. A neurological status revealed right facial and lid weakness, marked weakness of the right arm, especially the hand. A right Hoffman sign was present. Right corneal reflex was diminished. Deep reflexes were increased in the right arm. The abdominals were normal. Eccymoses about the legs were still present in faint traces. The operative scar was well healed.

October 1: He was up gradually. Laboratory status: RBC 4.01 million; WBC 6,400 with polys 46; lymphs 52; basophils 1; monocytes 1. Coagulation time: 2 minutes. Bleeding time: 1 minute, 40 seconds. Prothrombin time: 32 seconds (control 29 seconds).

About the middle of October the facial weakness and the paresis of the right arm were almost gone. At that time, he began to note involuntary twitchings of the fingers of right hand which persisted. His hematological status remained relatively stable. He was placed on phenobarbital 0.03 gm. three times daily, with marked improvement. Gradually he became more active and with the exception of two episodes when he felt "as if something dreadful was to happen in his head" he had no seizures and only rare involuntary twitchings of his middle and ring fingers when he was fatigued. There were no other abnormal neurological signs; the right brachiofacial paresis had completely cleared.

An electroencephologram report on December 3 showed: "moderately strong H-V response and recovery somewhat poor. Some random 5-7 p. s. activity which occasionally borders upon pattern formation." It was labelled "Borderline". It was decided to discharge this patient after a period at a convalescent hospital on small doses of phenobarbital. There was no indication that any surgical intervention would be of value in relieving the irritative cerebral focus so readily controlled by small doses of phenobarbital. There was a reasonable expectancy that it would clear as had the rest of the cerebral pathology.

Case II: R.P.D. Age 19. White male. Admission date: July 21, 1945.

History: Twenty hours before admission the patient noted a rash of pin-point reddish-purple spots on his legs. A few hours later he noted small purple swellings in his mouth and bleeding when he blew his nose. Gradually, new similar swellings appeared in other areas of the skin. Past history was negative for exposures to chemicals or drugs and there had been no previous episodes of hemorrhage or bleeding. There was no family history of blood dyscrasia. Physical examination on admission revealed as positive findings, several small hemorrhagic areas on the face. Dense areas of petechiae were scattered about the legs, arms, buttocks, chest and abdomen. There were hemorrhagic areas in the mouth along the gums and two small subconjunctival hemorrhagic areas in the left eye. There were small dry crackling rales in the left chest posteriorly. Spleen not palpable. Admission blood count was: WBC 7,360 with normal distribution; platelet smear revealed a complete absence of platelets; RBC 4.9 million; coagulation time 3½ minutes; bleeding time: patient continued bleeding after 1½ hours. Blood type A; Kahn negative. The patient was promptly transfused with no reaction. Diagnosis was purpura haemorrhagica. He remained relatively stationary and very few new purpuric spots appeared since admission and since 250 cc. transfusions on July 21, 22 and 27.

Pertinent data show that on July 23 the urine examination was normal. The RBC was 5.23 million; Hgb 100%; WBC 6,650 with bands 3%, segmented 58%, lymphocytes 34%, eosenophils 1%, monocytes 4%. The platelet count was 10,000/cu. mm.; sedimentation rate was 5 min./hr. On July 24 the platelet count was 18,000/cu. mm. On July 26 the platelet count was 24,000/cu. mm. The RBC was 5.23 million, Hgb 97%; the WBC was 9,100 with 73% segmented, 20% lymphocytes, eosinophils 1%, band 1%, monocytes 5%. Prothrombin time: 24 seconds (control 28 seconds). The next day the platelet count was 42,000/cu. mm. A urine examination was cherry red in color, 4 plus albumin and loaded with red blood cells. Prothrombin time was 24 seconds (control 28 seconds).

Seven days (July 28) after admission he complained of headache and blindness. Positive findings on examination revealed blurred optic disc margins, a left homonymous hemianopsia and mild nuchal rigidity. It was thought that a hemorrhage had occurred into the right cerebrum. Later that day the headache increased in intensity; he complained of photophobia and he vomited. Blood clotting time was four minutes; clot retraction was not complete after 48 hours; icteric index was 8. That night he vomited 200 cc. of coffee colored fluid. The next morning he appeared to be irrational, restless, blood pressure began to rise and pulse became depressed. An examination at this time revealed a totally irrational male with a left sided weakness, diminished to absent deep reflexes on the left, absent abdominals on the left and a stiff neck. The fundi showed blurring of the discs but no papilledema. Blood pressure was 170/80. A spinal fluid examination, after a non-traumatic tap, revealed: RBC 54,250 and WBC 11. The initial pressure was 600 mm. of water; the fluid was bloody.

For the first time, this patient was seen by the neurologist who found him to be stuporous, restless and uncooperative. There was a loss of tone to the left side of the face with a slight lag of the left side of the mouth. The discs were blurred bilaterally with about two diopters of papilledema but no hemorrhages. Pupils reacted to light; reflexes were depressed on the left; abdominals were absent on the left. The Babinski sign was equivocal bilaterally. There was flaccidity of the left arm and leg. He was transfused,

given hypertonic fluids and on July 30, nine days after admission, a splenectomy was performed. The next day the platelet count was 330,000/cu. mm.; coagulation time was 1 minute, 45 seconds; and bleeding time 3 minutes. RBC was 4.79 million, WBC 20,100 with 86% polymorphonuclears, lymphocytes 11%, eosinophils 1%; bands 2%; Hgb 91%. That night a lumbar puncture was performed. Initial pressure was 380 mm. of water; the fluid xanthochromic. The neck remained rigid but the stupor lessened.

The resected spleen was examined by the pathologist who reported: "a normal sized spleen, wt. 164 grams, measuring 11x8x3.5 cm., normally soft in consistency. Follicles prominent on cut surface. Microscopic examination showed hyperplastic follicles. There was only a small amount of phagocytized blood pigment. Thrombcytopenia produces no histological changes pathognomonic of the disease."

The patient improved, but was restless and continued to complain of a severe headache. On the 7th day post operative (August 6) he appeared rational but complained of blurred vision. A urine examination at that time was essentially negative. NPN shortly before this date (August 3) was 27 mgm; tourniquet test was negative. On August 11, 12 days post-operative, rigidity of the neck returned, spinal fluid was again under increased pressure, 320 mm. water and the fluid xanthochromic; Kahn was negative; colloidal gold curve normal. There were 32 RBC and 10 lymphocytes on microscopic examination. WBC was 13,100 with polys 86%, lymphs 10%, monocytes 1%, bands 3% Hgb 91%; platelet count 484,000/cu. mm.

On August 14 another spinal puncture was done and again the initial pressure was elevated, 340 mm. water. The neurosurgeon believed that there was a right cerebral blood clot and that intervention was indicated. On August 15 the right occipital cortex was exposed after trepanation and dural incision. It herniated into the opening and the bony opening was enlarged after the clot was reached with biopsy needle at 4.0 cm. depth in the occipital lobe. The clot was sucked out, the cavity visualized and the septum between cavity and temporal horn ruptured. Bleeding parts were coagulated and a small tantalum plate wedged into the bony opening. His post-operative condition was good. Examination on August 17 revealed no particular change.

A spinal puncture on August 21 revealed initial pressure 310 mm. and faintly xanthochromic fluid. Total protein was 75 mgm. %; cell count 7 lymphocytes; sugar 58 mgsm. %. He continued to improve and by August 23, his neck was supple and there was no headache. However, he complained of severe pain in his legs which was relieved by removal of spinal fluid until the pressure dropped to normal. On September 5 the discs were still hazy, reflexes in the left leg were still absent and he complained of parasthesias there. The left homonymous hemianopsia persisted. Gradually he continued to improve and by the end of October the left homonymous hemianopsia and what appeared to be a peroneal nerve lesion of the left leg were the only positive findings. The peroneal palsy was considered to be unrelated to the central lesion and to be of the idiopathic variety. The steady progress continued with the assistance of physiotherapy and his being up and about. When he was last seen by the neurologist on December 4 he found a residual left homonymous hemianopic field defect with a specific inferior quadrantic defect in the left eye. Superior margins of both discs were blurred and the vessels tortuous. There was a typical left peroneal nerve palsy with concomitant signs.

He was discharged to a convalescent hospital in good physical condition and contact was lost.

Comments

Case 1: R.H.E., illustrates an acute onset first mistaken for a septicemia, a hemiplegia with convulsive phenomena and spectacular recovery with only a residual irritative focus in the left cerebral cortex which was readily controlled by small doses of phenobarbital. In this connection, it is interesting to note that the electroencephalogram did not report a localized focus of activity which was so evident clinically.

Case 2: R.P.C., illustrated an even more dramatic recovery which required, in addition to splenectomy, neurosurgical intervention to evacuate an intracerebral clot. He, of course, presented many of the features of subarachnoid hemorrhage. The peroneal nerve palsy was considered unrelated to the purpuric picture. His eye findings were interesting and a visual field defect persisted but was not incapacitating.

Both cases prove the life saving value of splenectomy in acute, downhill cases of thrombocytopenic purpura. Both cases illustrate that even with apparently marked cerebral complications, judicious judgment and courageous treatment make the outlook brighter and such complications clear in greatest part. This is particularly illustrated in Case 2.

Summary

1. Two unusual cases of essential purpura haemorrhogica with cerebral complications are presented.

2. Both recovered after splenectomy and one required evacuation of an intracerebral clot.

3. Both had residuals of the vascular brain lesions; in neither, incapacitating.

4. Other than the hemotologic abnormalities, there was no pathognomonic organ pathology.

5. Splenectomy is a life saving measure and marked cerebral complications need not be a deterrent to such procedure.

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The Physician's Field Workers for Mental Health

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Physicians do not work single handed. They have allies, sometimes unrecognized, in the community.

Numerous studies indicate that 50 per cent or more of the patients seen by an internist or a general practitioner suffer from ailments which are either due to, or complicated by, psychological factors. Much has been written regarding the effects of frustration, indecision, and emotional conflict upon an adult person's digestive processes. Pediatricians are now as much interested in the emotional attitudes and habits of children as in the purely physical factors involved in feeding, growth, and general development. During the war an intensified research program has been conducted on the relation between psychogenic factors and recovery from surgery, as for example in amputation cases.

Because he usually sees the problem only after the breakdown occurs, the physician is too often impressed with the purely physical symptoms. He recognizes the background for the psychosomatic features, but usually it is too difficult to deal with, and moreover he does not have the time and not always the training which are essential to successful treatment.

Allies in Preventing Emotional Difficulties

The physician will be encouraged to know that in every community there are allies who have been prepared to render valuable assistance. Other professional groups are now working more intelligently toward the prevention of emotional disturbances in children and adults.

The teacher is an ally albeit not always recognized. In the old days she was trained only to teach academic subjects. Now her preparation consists more and more in the understanding of processes of growth and development in children. As this understanding increases, the teacher is better able to help children develop to maturity unhindered by parental antagonisms, failures in social adjustment with other children, or ill-advised vocational placements. Individuals free of these complications are better able to take their place as stable adults in family and community life. From time to time, they have worries which may produce physical symptoms, but they are more likely to know how to handle their difficulties before serious breakdown occurs.

The social case worker is an ally. Modern training in child welfare and family service stresses an understanding of the inter-relationship of emotional, social, and health factors in the development of a personality. These workers do not presume to have professional knowledge of physical symptoms and treatment, but they do understand the human tangles which lead to distraught emotions and in turn to physical malfunctioning.

Important in a social worker's course of

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training is a knowledge of his own limitations. He leans heavily upon the counsel of a physician and the help of a psychiatrist. Nevertheless a case worker is also of help to these professional groups, by dealing with individuals at the preventive and rehabilitative level.

The clinical psychologist is an ally. Many people find themselves in impossible situations because they are the proverbial square pegs in round holes. This is particularly true vocationally. Psychologists are trained in group and individual testing and in other methods of understanding the individual's potentialities and aspirations. Vocational counseling does not determine anyone's future, but it may help one discover his own interests and abilities. Planning based on such facts is more likely to bring self-confidence and a measure of success than is trial and error blundering.

The psychologist is also making his contribution in the understanding of the emotional and mental development of children, the readjustment problems of adults, and more recently, the mental health aspects of old age. Eventually, the psychologist will join with the physician and psychiatrist in contributing to an understanding of the new field of geriatrics.

The minister is an ally especially if he has had modern training in counseling. Several theological seminaries now include as a regular part of their course an interneship in a mental hospital or at a guidance clinic. This experience gives the clergyman a better understanding of the individual's emotional problems. Through such understanding and with the use of the elements of faith and worship, which are a part of his tradition, he can help persons regain confidence whose plans have been shattered.

Ministers do not attempt to counsel persons who are seriously disturbed. Part of their training is to know when to call on the expert. But the minister with modern training does understand general emotional needs which enables him in his day by day parish contacts to give help to many people.

Business and industrial personnel managers are allies. In the old days they were trained simply to lay out the job and hire the workers. Now their objective is to achieve a harmonious adjustment between the person and the job, and within the work group. Formerly, the emotional needs of individuals had to be satisfied away from work. Now, more and more attention is given to the recognition of the individual employees. He is less a cog and more a person in a big corporation. Furthermore, as the stability of the individual and the morale of the work group increase, so also does the production output of the company.

There are many more allies. A fuller list would include the specialist in parent education, the trained probation worker, the counselor in the rehabilitation service, in the employment service and in the Veterans' Administration.

Public health nurses, the county and state health department, and other professionally trained health workers, obvious co-workers with the physician, are not included in our list. Rather, we have stressed the supporting service of professions not necessarily identified with health, but whose work is clearly related to mental health. As the physician sees the importance of emotional stability in the prevention of illness, he will seek help among the allies whose work is here indicated.

The Physician's Role

The physician himself has an opportunity and a responsibility in this development. He is a prestige person in every community—oft times of the very highest prestige. His friendly interest in his allies can be a great boon to improvement in education, in family life, in religion, in personnel methods of industry, and in the other fields of human adjustment. His endorsement of a community child guidance clinic, or of a vocational counseling program in the schools, or of professional training for the welfare worker will count for much in the improvement of community life. Through such measures, the general quality of living increases and the emotional adjustment of individuals and families becomes more secure. These conditions are relevant to the physician's desire to prevent mental and physical illness and to speed the patient's recovery when it does occur.

Neurosis—A Progressive or Self-Limiting Disease?

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Neurosis, as Freud proved 50 years ago, is an illness of the unconscious. In unraveling the Gordian knot of neurosis we find with greatest regularity an unconscious wish, an unconscious inhibition and an unconscious defense mechanism. The neurotic end-results are deposited either in neurotic signs and symptoms or in "personality-difficulties."

The question arises as to the fate of untreated neuroses. What happens to them? Do they burn themselves out automatically? Are spontaneous "cures" effected? Is neurosis basically a progressive or self-limiting disease?

Clinical experience proves that neurosis is, unfortunately, a progressive illness. An untreated neurosis deteriorates as the years pass by. Symptoms may change and be shifted from one organ to another; depressions and worries attach themselves to different rationalizations but the neurotic basis remains. Not only does it remain, but the ability of the personality to absorb like a sponge libidinous-aggressive conflicts, decreases and the general picture worsens.

Every neurosis contains a quantitatively different amount of self-damaging tendencies. This psychic masochism can, as life goes on, be placed at different points. Confronted with either self-created or purely external painful or unhappy situations, the neurotic spends his wish to suffer, with the result that he has, temporarily, less symptoms and feels better. He might be compared to a drunkard who needs a specific dosage of alcohol every day; at what bar he gets it, is unimportant. Freud mentions apparently incurable cases of severe obsessional neurosis who lost their symptoms subsequent to organic disease, loss of money, or after other external unhappy events occurred.

If we do not take into account the exchangeability of neurotic manifestations and overlook the static basis of neuroses we may be led to erroneous conclusions. Specialization of medical science bolsters this fallacy. A girl of 18 may develop a "nervous stomach" and after treatment by a gastroenterologist is helped. Two years later she may suffer from migraine-like headaches and a neurologist is consulted. In the meantime she married and divorced two husbands. After her return from Reno she has severe abdominal pains; appendicitis is suspected; a laparotomy is performed but the appendix proves to be normal. A few months later the headaches set in again. She consults another neurologist. Later the patient complains about all sorts of hypochondriacal ills, and consults in rapid succession a gynecologist, an internist and a sinus specialist. All her life she remains a patient of "universal medical science," a non-diagnosed and not improving neurotic.

In determining whether or not a neurosis deteriorates, one has to take into account the development of this illness. Neurosis is the petrification of an infantile conflict which is repressed. The infantile conflict itself, without psychiatric help, is indestructible. Every neurosis, which is latently present from early childhood, manifests itself first in form of depressions and fears. That does not mean that these are genetically the first depositions; they are merely the first visible ones. The "personality" *per se* absorbs the conflict first. Only after this "sponge is saturated", do symptoms and signs appear which the conscious Ego treats like a foreign body. In other words, the "queer" personality precedes the "queer" neurotic symptoms.

Deterioration of a neurosis is difficult for the patient and the non-specialized physician to trace because there are years in the life of a neurotic when, seemingly, everything is "alright". The process of deterioration reveals itself only if one takes into account an overall picture of the personality. Then, and then only, does it become apparent that the neurosis of the thirties is worse than in the twenties, and so on in direct progression. If one meets a man with severe neurotic symptoms today, loses sight of him, to encounter him two years hence and at that specific time the person has "shed" all his neurotic difficulties, a fallacy is possible in the judgment that the man "overcame" his neurosis. In

probing deeper, however, one finds, for instance: that in the meantime the man had gone through an unhappy marriage and divorce which, since unconsciously self-created, consumed unconsciously, for the time being, his psychic masochism and therefore he feels "fine".[1]

I should like to demonstrate the deterioration of untreated neuroses by the example of "overaged" bachelors.[2] The elderly bachelor in the early forties or fifties rationalizes his being unmarried with lack of opportunities, time, money and what not. No one believes these reasons and even the psychologically untrained observer calls him "queer" which is a popular synonym for "neurotic". This individual often acquires the reputation of being a "wolf" since, typically, he has a series of transitory affairs. His potency seems thus established; it is only marriage he avoids. Everybody tries to push him into marriage, until one day, to the surprise of everybody, the man becomes engaged and married. The man's behavior gives the impression that he is at last doing the "normal thing". If one analyses such a repentent enemy of marriage, it becomes clear, very soon, that behind the facade of bowing to normalcy he married because his neurosis had increased and asked for a holiday in form of an exaggeration. His aversion to marriage was a symptom of his neurosis, and paradoxically, at the same time, a means of protecting him against the full bloom of his latent neurosis. Here is a typical example:

Mr. A. consulted me because of marital difficulties. He was good-looking, in his early forties, with the charm and cynicism of a gamin. He explained his constant conflicts with his wife very simply: He was a "born bachelor," who did not marry, but was married by his wife. He had warned her but she insisted, "and what can you do against a woman's determination?" He spoke disparagingly of women in general. They were inferior creatures with no brain except for intrigue. I asked him about his conflicts with his wife. His answer: "Oh, she is terrible; she behaves like a governess, makes constant rows, is hysterical and overbearing." "How long did you know your wife before marrying her?" I wanted to know. "A year or so. But never mind, she changed completely. She told me then that she would give me back my freedom if our marriage didn't turn out. Well, it hasn't in three years, and now she refuses to divorce me." Asked what his wife had to complain about in him, he admitted that he "made some little mistakes." For instance, love letters from other women and his replies came "accidentally" into his wife's hands. He was, he constantly reiterated, a "born bachelor," and considered himself justified in having extra-marital relations. Now, unfortunately, his wife had "material" against him, and that "complicated the matter."

A conversation with Mrs. A., a smart-looking woman appearing not more than thirty (although she was forty), revealed that she considered her husband "a terrible liar, in every respect unreliable, mercenary, and good-for-nothing." She denied that she had forced him to marry her and pointed out the good influence she had on him. She conceded that there were rows, but shifted the responsibility upon him. "I never made scenes before I discovered those letters, but afterwards I wanted to know the truth." According to her, she fought only when her husband came out with "dirty lies." "If he would only confess, I would forgive him, but he lies like hell." Asked why she insisted on having all the details, she answered that she was afraid to meet people any longer, for she didn't know which were his girl friends and who knew about them. She was afraid of becoming a laughing-stock. Full of fury, she accused her husband of being a "block of ice." "Of course, he has the advantage since, when I react emotionally, he calls me hysterical."

From the life story of Mr. A. it could be gathered that his beautiful mother had had the greatest influence on him, his father having been away often during his formative years. The patient emphasized the beauty of his mother and it was evident that in his pseudo-charm he identified with her. Soon it became apparent that he had unconsciously a strong feminine identification. This tendency he counteracted unconsciously with the pose of a Casanova and with intellectual disparagement of women. Incidentally, he gave as one of his reasons for allowing himself to "be married by" his wife, his desire to break away from his "polysexual life." Without consistence, he stated on the other

hand that he preferred his bachelor life. In any case, it became clear that all the "bad qualities" he accused his wife of having (especially those of an overbearing governess) were exactly the ones which could fulfill his inner wish of being overwhelmed in a feminine way. Unconsciously there was in that layer for them a complete reversal of the man-wife attitude. He played inwardly the role of the woman; she, that of the man.

Interestingly enough, the patient was sexually uninterested in his wife. He attributed his lack of interest to two facts: first, that she was not his "type" and, second, that her constant rows made sexuality impossible. The man was very potent but wanted to be seduced by his wife. She, however, played coy and was rather passive in sex. A chief objection to his wife was that, in a previous relation, she had actually seduced a man, a deed which she had mentioned to him as a practical joke when their acquaintance was slight. The patient dwelt constantly upon this incident, resulting in more reproaches and more refusals in sex and money. Despite the fact that his wife understood vaguely that her husband wanted to be seduced, she felt an inexplicable inhibition against "going back to the old times."

We see that the man unconsciously wanted to be overwhelmed in feminine identification —his wife was aggressive. He wanted, furthermore, constant hysterical rows since they gave him the feeling of being mistreated—she also gave him that satisfaction. However all these satisfactions were on the unconscious level. Consciously, he suffered "like a dog," to quote the patient. His wife provided for him only unconscious pleasures on a characterological level; their sexual life was highly unsatisfactory. There the inner defense on his part set in. What he really wanted was not genital satisfaction. He had used this satisfaction, in previous relations, to a great degree to establish the alibi of being a "he-man." Basically his being mistreated satisfied his sexual needs in a masochistic way. But his constant complaints against his wife represented his unconscious defense. Even in this defense he smuggled in his old wishes; by refusing pseudo-aggressively sex and money, he provoked more aggression on her part and started once more the vicious cycle of being unjustly treated.

The strange part in the unsolved oedipal relation was that his wife did not represent his "type," whereas women he had known in previous relations did. Obviously this was because in marriage his defense had to advance further in protecting him against his original wish, since it was easier to identify unconsciously his legitimate wife with his mother than girls of indifferent and transitory affairs. Therefore the defense had to go a step beyond—avoidance of his type. Even in his previous transitory affairs, however, it became obvious that our patient was not the great seducer he pretended to be. On the contrary, these women had to seduce him. By means of his constant infidelity he tried in vain to prove to himself that he was, not passive, but aggressive toward women.

It might be asked how it happened that a man who had escaped marriage until he was forty should finally have succumbed to it. The answer is that his original wariness against marriage represented an unconscious desire to avoid greater neurotic troubles, a form of self-preservation of the Ego against greater inner danger. But, as is true of old bachelors when they marry, as they often do in their forties, his neurosis increased with age and he eventually bargained for greater trouble and got it. His increasing craving for neurotic pleasure was also visible in his unconscious concocting of the situation in which his wife found the incriminating letters. Neurotics at that age want to repeat their neurotic repertoire one hundred per cent; innuendoes no longer satisfy them.

If we ask why this man married at all, we see that it was because his neurosis had increased, and Mrs. A. presented him with a first-class opportunity to reel off his neurotic pattern.[3]

It is amazing to what degree the progress of an untreated neurosis is independent of reality, or more precisely, how reality is just used as a tool for the neurosis. A tragic example of this fantastic tendency is the following:

Many years ago, I was requested by the family of a compulsive neurotic to treat this man who spent all his time washing his hands. (Wash-compulsion). Of course I wanted to see the patient first but he declared himself

an enemy of psychoanalysis and refused even to see me. A few years later, the patient, a Viennese, was summarily arrested during a Nazi pogrom and placed in a concentration camp. There he behaved most strangely; the same man who had washed himself all day stopped washing altogether. At that time the Nazis were still in their beating-torturing stage; the outright killing and extermination came later. One day the S. S. guards decided to "teach" the man cleanliness; on an extremely cold winter day they stripped him in the open yard and kept pouring buckets of water over him for several hours. The victim died of pneumonia.

The reasons which prompted this man to give up washing can only be vaguely surmised. Clinical experience with analysed cases of compulsive washing proved that these neurotics constantly repress the wish to play with feces as end-result of their infantile conflict. This wish, having for these people a "sexual" connotation, is counteracted by a severe reproach of the inner conscience, with the result that in consciousness the opposite tendency appears; namely: Compulsive hypercleanliness. Since both, unconscious wish and unconscious reproach, never end because of inner ambivalence, the wash-compulsion is perpetuated. In the concentration camp the man obviously appeased his severe inner conscience with the objectively unhappy situation, with the result of "acquiring" the inner permission to indulge in his dirt-mania. At the same time he shifted the punitive factor of his inner conscience to the Nazis. There is reason to assume that he would have survived had he behaved differently. At that early stage the Nazis released non-political prisoners who could produce a visa to a foreign country, which his parents were able to secure.

As a third example I present the case of a young lawyer whom I treated for a few months, 15 years ago. The patient entered analysis because of psychogenic impotence based on hysteria.[4] After a relatively short time he was able to perform intercourse successfully for the first time in his life. Immediately afterwards he stopped treatment declaring himself "cured". I protested in vain, pointing out that he fooled himself completely about the depth of his neurosis. I advised him to continue treatment lest he suffered a predictable relapse. The man could not be influenced even so far as to discuss the experiences with similar cases. He felt cured and "that was that". Some months later I received his marriage announcement. Shortly after he consulted me in great excitement informing me that his wife suffered from vaginismus and had been treated surgically. The patient was aware that this condition is the manifestation of a neurosis and tried to convince his wife to enter psychoanalysis. Surgical treatment of vaginismus was, as could be expected, without effect. His wife refused to be analyzed and took her condition fatalistically. The patient, too, accepted the situation with remarkable stoicism. What it really amounted to was that neither of them wanted genital sex. Every attempt at explaining this to the patient failed, though he admitted that even before marriage he had had the "vague feeling" that his wife was "sexually not too interested." What he did not admit was that this impression was precisely the reason for his choice. Fourteen and a half years later I met this man on the street. He was still married to his wife. She remained virgin because of her neurosis but still refused to be treated. He accepted her dictum without complaint and was still "faithful" to her. His external circumstances were poor, his wife earned more than he did. He often remembered my advice to continue treatment. Perhaps, he remarked hopefully, he would even do so—one day....

Neurosis is a dangerous and progressive disease. Symptoms may be exchanged, but the condition advances; the masochistic stakes increase; the overall picture deteriorates. To hope for spontaneous cure in neurosis is as senseless as to expect the Niagara Falls to flow in the opposite direction.

251 Central Park West.

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Neurological Clinical Pathological Conference

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CHARLES D. ARING, M.D., *Editor*

Case Number 172366, 195994

Presentation

(The following two cases are comparable from the neurohistopathological viewpoint).

A 3 year old colored female had been well and as usual had been taken by her mother to a day nursery on the morning of July 8, 1944. At the close of day a nurse told the mother that the child had been vomiting intermittently throughout the day. She refused supper, but took some milk and vomited. She had also vomited on the morning of July 9.

The child was brought to the Out Patient Department of the Cincinnati General Hospital on July 9 to keep a regular appointment for the inspection of a repaired abdominal hernia, which had been done two years before. On the way the mother noted that the child was sleepy. On arrival at the Surgical Clinic the child would not respond and shortly afterward had a generalized convulsion. She was brought to the Receiving Ward at 9:50 A.M. On July 8 Cincinnati's maximum and minimum temperatures were 95 and 68, on July 9, 99 and 70 degrees.

The child had had a normal birth at this hospital. She had developed normally and was "bright" mentally. She was one of five children; all were well. The only illness had been mumps four months before this admission, and measles three months before entry. She had had smallpox vaccination at the age of ten months, and diphtheria immunization.

In the Receiving Ward the patient's temperature was 99.4° F. rectally. On reaching the ward, the child was convulsing and the temperature was 100.4°. Fit succeeded fit until 4 A.M. of July 10, despite seven intramuscular injections of sodium luminal, each of 1 grain. She was in coma between seizures. The attacks were prolonged usually lasting from five to ten minutes, with a short period of coma between attacks. Most of the seizures were Jacksonian in type, beginning in order of frequency in the right side of the face or the right upper extremity or the left side of the face. The convulsion would usually spread to an extremity or the extremities of the same side. Sometimes there would occur alternation of sides from attack to attack.

Formal examination was not revealing. The deep reflexes could not be obtained between the seizures, or in the deeply comatose state that followed the cessation of seizures. In the single recording of the state of the pupils, there was said to have been no response to light. Sucking movements of the lips occurred on cleansing the mouth.

She never aroused from coma and died after 37 hours in the hospital, despite symptomatic supportive therapy.

Laboratory Data: The temperature which was normal in the Receiving Ward rose to 103° five hours later and to 105° seven hours after admission. The pulse rose with the temperature, and became irregular; respirations then became fast and were labored. Periods of apnea were noted twelve hours after entry to the hospital. With the use of tepid sponging, fans, and icebags, the temperature was reduced to 102.5° twenty-four hours after entry; it again rose rather gradually to a high of 105.8° two hours before death.

Absorption of fluids administered beneath the skin was poor throughout the hospital stay.

A single urinalysis revealed sugar and acetone. The white blood count was 18,000 on two occasions, the lymphocytes were 50%, the hemoglobin 11 grams. The blood Kahn was negative. Blood culture was negative. Blood sugars done on the successive days were 200, and 153 mg.% respectively, the CO_2 combining power on the final day was 34.5 vols.%. Blood culture was negative.

The first spinal puncture revealed a pressure of 185 mm. of water, and 10 cc. of bloody fluid was obtained, containing 32,000 red blood cells and 150 white blood cells per cmm. The tap was repeated ten hours later when the patient was having labored respiration with periodic apnea. Clear cerebrospinal fluid was

obtained under a pressure of 390 mm. of water, 250 red blood cells and 29 white blood cells per cmm. were counted, the protein was 200 mg. the sugar 132 mg. and the chloride 774 mg.%. This cerebrospinal fluid was obtained after the administration of three infusions containing 5% glucose solution, the last having been begun 1¼ hours before the lumbar puncture.

A 5 month old white female infant was admitted to the hospital on August 12, 1944, at 7:31 A.M. She had been irritable, restless, and crying for the most of three days and nights. On the evening of August 10th a physician had examined the baby to find the temperature normal, and the remainder of the examination normal also. Cincinnati was in the midst of a prolonged heat wave, the maximum and minimum temperatures were 98 and 75 degrees on August 11, and 98 and 71 degrees on August 12. The home was not an unusually hot one.

On the morning of August 12 the child developed irregular, shallow, and labored respirations which resulted in rather immediate admission of the patient to the hospital, where death occurred at 8:05 P.M.

On admission, there were coma and cyanosis, and respirations were about 150 per minute, the pulse rate was 150. The temperature was 108° F. The extremities were cool while the trunk was hot. The feet were plantar flexed into an equinovarus position.

The examination could not be thorough since the condition of the child was precarious. Oxygen was begun as was an intravenous drip of 5% glucose in saline. Vigorous therapeutic attention was maintained for the less than 12 hospital hours of life.

There was a tendency for the temperature to be maintained around 107 to 109°, and continuous attention and effort were required to keep it down to 104° (alcohol and ice sponges, electric fan, fluids, stimulants).

Several periods of "spasm resembling tetany" were noted by the nurses. By noon of August 12 the extremities felt warmer. At 2:55 P.M. it was noted that the patient was "apparently having chills," and sponge bathing was then discontinued. The temperature was then 102.6°. At 5 P.M. constant jerking movement of the left leg was noted. Again the extremities were cold, and the temperature was 106.8°.

Extreme cyanosis remained throughout the hospital course, and respiration was always labored. Long periods of apnea set in at 3:40 P.M. The child was never conscious in the hospital.

Laboratory Data: The urine was normal. The white blood count was 22,400 with a lymphocytosis of 56%. The hemoglobin was 9 grams. Blood culture was negative, and stool culture negative for typhoid and dysentery. The blood CO_2 combining power was 27 vols.%.

A lumbar puncture revealed a pressure of 50 mm. of water, 6 white blood cells and 120 red blood cells per cmm. were counted, the protein was 19 mg.%, the sugar 90 (after glucose intravenously).

Differential Diagnosis

Dr. A. Ashley Weech: An initial comment is in order on the kind and number of diagnostic studies which were carried out on these patients. Both children were critically ill so that symptomatic treatment demanded first consideration. Survival times were only 37 hours and 12 hours respectively. Under the circumstances it is assuredly unseemly to ask why additional data were not requested from the laboratory. But, because retrospective recognition of omissions can improve clinical management in the future, it is proper to bring the subject up. Understanding of the disorders which these patients exhibited would be enhanced if there were a more complete picture of the electrolyte pattern in the blood. In particular, we should like to have had determinations of serum pH and serum calcium.

An early comment is also in order on the possible diagnostic import of several of the clinical findings. In Case 1, the convulsions were described as being Jacksonian in type. I do not think any localizing significance can be attached to the fact that there was a "march" to the convulsions. Likewise, abnormal neurological responses in the wake of convulsions are notoriously unreliable diagnostically, and I think no significance should be given to the absence of deep reflexes in the intervals between seizures. In Case 2 the

single urinalysis revealed sugar and acetone. There is no other evidence to suggest that the child was diabetic. I shall therefore assume that the glycosuria was alimentary and resulted from the therapeutic administration of glucose solution. Acetonuria in children who have been vomiting is to be expected and at this time requires no explanation. The observations on the spinal fluids from both patients are chiefly of negative value. They serve to exclude the ordinary infectious types of meningitis and they have no positive value to support a diagnosis of encephalitis. The fluid from both patients showed red cells. The finding may or may not have significance. In Case 1, the initial tap yielded bloody fluid with a proportional relationship between red cells and white cells so similar to that in the circulating blood, as to make it probable that this was a "bloody tap". The second puncture was done only ten hours after the first. We must recognize the possibility that the findings in this fluid may have resulted from irritation of the meninges by blood introduced by the trauma of the first puncture.

We shall now turn to a discussion of diagnostic possibilities. Since the children exhibited fulminating types of illness associated with fever and symptoms of central nervous system origin, it is proper to ask whether or not they were victims of encephalitis. The word "encephalitis" is often used loosely and improperly to embrace forms of cerebral irritation which are not infectious in origin. Such loose meaning is not intended here. I am considering types of disease caused by the presence of neurotropic virus in the nervous system.

These cases occurred in the summer season. This fact and the rapid course are consistent with what might be observed in the St. Louis type of encephalitis. But, we also should have expected a significant increase in mononuclear cells in the cerebrospinal fluid and an increase was not found. Moreover, it is impossible to believe that two cases of St. Louis encephalitis could have broken out in a community in which there was no epidemic among the adult population. Indeed, I do not know of any type of encephalitis to which these or even more serious objections are not applicable. There remains the possibility of infection with a still unidentified virus. No justification exists for the belief that all types of virus capable of attacking the nervous system have now been isolated and identified. The possibility of infection with an unknown virus can therefore not be excluded. Nevertheless, it is more reasonable to inquire whether other mechanisms, non-infectious in nature and about which there is positive knowledge, could have produced the clinical disturbances exhibited by these patients.

In the histories emphasis has been placed on the extreme summer heat prevailing at the time. Could the children have suffered from heat stroke? In Case 1 it is unlikely that environmental conditions played more than a secondary role. Infants are not infrequently upset by extreme and protracted summer heat but this child was past the age of infancy. In older people the incidence of heat stroke increases with advancing years and is most common after the age of 60. This child was colored and the colored race tolerates heat better than the white. Finally, the record suggests that there was no fever at the time of the onset of convulsions. In Case 2, which occurred in a 5 month old white infant, it is possible to assume that the irritability, restlessness, and crying during three days and nights were symptoms of heat prostration which culminated in heat stroke on the morning of admission. Too little is known of the effect during infancy of heat uncomplicated by other disturbing factors. It will be helpful to discuss several of these factors which are capable of producing symptoms of cerebral disturbance before concluding that environmental temperature played more than an accessory role in either case.

Probably closely related to the cerebral symptoms of heat stroke are the convulsions which often attend the onset of acute infections in young children. Wegman[1] has provided evidence from animal experiments to support his impression that such convulsive seizures are especially prone to occur when the rise in body temperature is abrupt. Although fully supporting data from the laboratory are not available, we shall note here the fact that rapid rises in body temperature are associated with rapid increases in respiratory rate and the possibility that hyperventilation of the lungs with ensuing alkalosis results therefrom. This point is impor-

tant, for it may furnish the link of similarity between the two cases under discussion today. In any case, Yannet and Darrow[2] have shown by chemical and histological observations that rapidly induced fever leads to demonstrable changes in the brain, namely, a loss of water without accompanying loss of electrolyte from the cerebral cells. At this point in the discussion, we shall merely take note of the fact that circumstances which disturb the water-electrolyte pattern of body fluids are capable of evoking cerebral symptoms and producing demonstrable cerebral change.

Doctor Rapoport, in the Children's Hospital Research Foundation, has recently become interested in a condition which he calls, "the post-acidotic syndrome". It is well known that children with severe diarrhea lose from their bodies large quantities of electrolytes and water and develop acidosis. Replacement therapy involves the administration of sodium bicarbonate and of fluids which contain the salts normally present in extra-cellular body water. It is not so generally appreciated that the disturbance is also accompanied by a loss of electrolyte from body cells and of lime salts from bone. This latter loss seems relatively unimportant during the acute phase of the illness when therapy must be directed primarily toward relieving dehydration. However, during convalescence there comes a time when recovering cells are avid to regain the salts which were lost. The essential ingredients of intracellular fluid and bone are withdrawn from the plasma. Potassium, inorganic phosphate, phosphatase enzyme, and calcium fall to subnormal levels in the serum. Among the variety of symptoms which ensue, convulsions, sometimes suggestively tetanic in nature, are prominent. The finding in several instances of xanthochromic spinal fluids is evidence that the reaction is attended by demonstrable change in the central nervous system. In the post-acidotic syndrome, then, we have another example of an association between a disturbance of the water electrolyte pattern of body fluids and manifestations of cerebral damage.

Several additional examples of this kind of an association can be mentioned briefly. Hyperventilation with loss of carbonic acid from the blood and the development of cerebral manifestations of alkalosis can result from a variety of causes. Sometimes the stimulation of the respiratory center is due directly to disease of the nervous system but more often it results from extraneural disorders such as laryngotracheobronchitis and pertussis. We have recently observed an infant who, during the treatment of severe diarrhea, received too little sodium chloride. Hypochloremia with alkalosis ensued and the infant died. Autopsy disclosed subarachnoid hemorrhages. As a final example we may mention the hypocalcemia of vitamin D deficiency which leads to tetanic convulsions; under these circumstances xanthochromia of the cerebrospinal fluid has also been observed.

It seems likely that the symptoms presented by the two patients whose histories have been recounted, resulted from a combination of the foregoing factors.

The first patient, the 3 year old colored girl, vomited all day on the 8th of July. She vomited again after supper and again the following morning. So much vomiting must have caused hypochloremia and dehydration. In consequence thereof the child became drowsy and developed the convulsions of alkalosis. The convulsions led to marked increase in the production of body heat. The environmental temperature and humidity prevented adequate loss of body heat and the child's temperature began to mount rapidly. The respiratory rate increased with the fever. Thus, hyperventilation augmented the already existing alkalosis. The appearance of intermittent periods of apnea indicates over-stimulation of the respiratory center. As I see it, these are the mechanisms responsible for the symptoms in this patient. The mechanisms may have led to subarachnoid hemorrhage, to shrunken cerebral cells and to cerebral edema. In any case, the primary disorder was the disturbance of water and electrolyte balance.

The second patient, the 5 month old baby girl, had been irritable, restless, and crying for three days. The cause of these symptoms is unknown. Perhaps extreme environmental heat was a contributing factor, but that it was not the only factor is attested by the fact that the phase of acute illness with hyperpyremia began in the early morning when environmental temperature was relatively low. One suspects the presence of in-

fection, perhaps in the respiratory tract. The respirations rose to 150 per minute and were described as labored. A low CO_2 combining power was found. The finding might speak for hyperventilation alkalosis with tetany since the feet were held in the position of pedal spasm.

In summary, one cannot in these cases exclude the possibility of infection with some unknown type of virus. Nevertheless, it is more likely that death came primarily from factors which disturbed the water-electrolyte pattern of body fluids and secondarily from the effect of this disturbance on the central nervous system.

Dr. Samuel Rapoport: I can add little more to Doctor Weech's discussion and therefore shall confine myself to a few remarks concerning the significance of the chemical findings.

The lowered CO_2 combining power, found in both cases, does not in itself signify the presence of a metabolic acidosis, and neither the clinical picture nor the other laboratory data indicate any mechanism for such an acidosis. Diarrhea, the most frequent cause of acidosis in infancy, was not present. Diabetic acidosis might fleetingly be considered in the first case on the basis of the findings of sugar and acetone in the urine; nothing in the course fits very well with this diagnosis. The normal non-protein nitrogen in the blood does not support the assumption of a renal disturbance as a cause of a hypothetical acidosis. On the other hand one may explain the lowered levels of bicarbonate in both patients on a basis of a respiratory disturbance. Irritation of the respiratory center with consequent hyperventilation results in a tendency toward an alkalosis of respiratory origin, with a lowering of the bicarbonate as a secondary compensatory change. In man, this compensation of the tendency to respiratory alkalosis is highly efficient, so that usually normal or only slightly elevated pH values are found.

It is of interest to speculate on the status of the electrolyte balance of the patients, in view of a story suggestive of dehydration. Usually dehydration is associated with losses not only from the extracellular compartment as a whole, but also with decreases in the concentration of electrolyte. This type of dehydration, where the loss of electrolyte tends to exceed that of water, is characteristic of enteritis and diabetes. Another type is characterized by loss of water without proportionate electrolyte. Such a condition may occur when large amounts of water are lost as vapor from the lungs as in hyperventilation or under circumstances in which the amount of nitrogenous waste products is large compared with the amount of fluid available to the kidney for excretion. Then, the kidney excretes nitrogenous products in preference to the electrolytes. In either case hyperelectrolytemia of very marked degree may ensue.

In the absence of simultaneously determined values for chloride and CO_2 in the plasma which might permit a reasonable estimate of the total electrolyte concentration, one may attempt by highly speculative and indirect reasoning to arrive at this value from the level of the chloride in the spinal fluid which was determined in Case 1. It was 137 mEq/L. One may remember that as a rule the chloride is higher by about 20 mEq/L in the cerebrospinal fluid than it is in the plasma. The blood CO_2 was about 16 mEq/L. Together these two anions may have accounted for about 133 mEq/L in the plasma. This value, while not extraordinarily high, gives some indication that the water loss might have exceeded the electrolyte loss in this patient.

The plasma calcium in Case 1, determined on the second day of the hospital stay, was found to be 9.6 mg. per 100 cc. On the face of it such a finding seems to exclude tetany as the cause of the convulsions. However, it may be recalled that in many cases of parathyroid deficiency, symptoms of tetany occur in the presence of similar calcium values. The level of calcium certainly is not the only factor concerned in producing tetanic manifestations. Fever is *one* modifying factor increasing the tendency to tetanic convulsions; hyperventilation is another. By itself hyperventilation of short duration may produce tetany without any change in the plasma calcium concentration. Lowered calcium values are found in patients with prolonged hyperpnea of central origin.

Hypocalcemia with its attendant convulsions, and hyperventilation, each may produce vascular damage in the brain. Several cases

of hyperventilation of unknown origin, have lately been seen in the Children's Hospital, in whom the cerebrospinal fluid contained an increased amount of protein. In the plasma a relatively high pH was found together with extremely low values for bicarbonate and for the tension of carbonic acid, indicating that hyperpnea was the primary cause of the electrolyte disturbance. These cases exhibited hypocalcemia of marked degree with levels as low as 6 mg. per 100 cc. Hypocalcemia without hyperventilation also has recently been observed following the severe acidosis of enteritis in infants. These patients in the "post-acidotic" phase during recovery are prone to develop, among other manifestations, convulsions which appear referable to their hypocalcemia. In several of such patients xanthochromic cerebrospinal fluid was obtained. In two or three cases massive intracranial hemorrhage occurred, apparently related to the tetanic convulsions and the hypocalcemia. In two cases of tetany on the basis of rickets xanthochromic fluid was observed following convulsive episodes. One might mention that the last two cases made uneventful and complete recoveries.

Dr. Eugene B. Ferris: What is the mechanism? It seems to me these patients got sick for some reason. Do you think that the alkalosis of tetany will be primarily responsible for the changes in the brain or will the changes in the brain be responsible for the tetany and hyperventilation?

Dr. Rapoport: The origin of this hyperventilation is obscure. If not the result of some pathologic process in the brain, it may be thought to arise on the basis of reflex irritation of the respiratory center. Such apparently is the mechanism of the hyperpnea in patients with laryngotracheobronchitis, which show inflammation of trachea and bronchi without impediment to the respiratory exchange.

Discussion of Pathology

Dr. I. Mark Scheinker: The gross examination of the brain of the older child disclosed swelling of the white matter of both hemispheres which narrowed the lateral ventricles. The brain of the younger child revealed congestion of the pial veins and there were numerous small petechial hemorrhages, chiefly in the white matter.

Microscopically the alterations were widespread in these brains. In that of the older child there was tremendous distention of the perivascular spaces, which harbored much serous fluid and a small number of gitter cells. As a rule the transudation of serous fluid was confined to the Virchow-Robin spaces. Occasionally, however, a disruption of the limiting glial membrane had occurred with the leakage of serous fluid into adjacent parenchyma. A narrow strand of nervous tissue proximal to the blood vessels showed myelin sheaths in all stages of degeneration (irregular outline, globular swelling, or complete loss of stainability). The nerve fibers suffered correspondingly in the same perivascular areas (swelling, globule formation, splitting, and reduction in number of axones). The glia participated in the process chiefly by regressive change (amoeboid degeneration and clasmatodendrosis).

There was an overwhelming predilection of the pathologic process for the vicinity of medium-sized and smaller veins of the subcortical and central white matter, with the tendency for the change to follow their course. In the case of the older child it is significant that the blood vessel walls did not reveal definite structural change. Except for moderate dilatation and engorgement, the larger blood vessels and the adjoining parenchyma were normal. There were no signs of inflammation; the leptomeninges and the cortical gray matter were normal.

What was grossly punctate hemorrhage in the brain of the younger child was found to be perivascular hemorrhage presenting about tremendously congested small veins. Most of the smaller blood vessels, especially the veins and capillaries in the white substance were tremendously distended. Maximum distention and stasis were accompanied by degenerative changes of complete necrosis of the vessel wall and therefore with increased permeability for blood. In those areas in which the accumulation of extravasated blood was especially pronounced, disruption of the limiting glial membrane had occurred. This resulted in diffuse rarefaction and disorganization of the nervous tissue.

The majority of these hemorrhagic lesions

were found in the centrum semiovale; they were also present in the subcortical white matter of different lobes. The cortical gray matter appeared fairly well preserved except for the presence of a moderate degree of edema. The leptomeninges were distended and the large blood vessels were congested. No signs of inflammation could be detected.

We have recently studied the brain of another child, aged one month, who lived 13 days after the onset of vomiting and diarrhea, the latter persisting until death. There were multiple thrombi in many of the tremendously distended veins and capillaries. No structural change in the vessel wall could be detected. The parenchymal alterations were more marked in this third case, and were characterized by disseminated areas of necrosis contiguous to the occluded veins.

The histopathologic findings in these cases displayed different phases of functional circulatory disturbance, characterized in Case 1 by an increased permeability of the vessel wall for serous fluid, in Case 2 by increased permeability for red blood cells as well, and in Case 3 by thrombosis of small vessels in addition to the increased permeability. The alterations of the nervous parenchyma were secondary to the vascular changes and consisted in Case 1 of perivenous tissue liquefaction, in Case 2 of petechial hemorrhage, and in Case 3 of focal areas of necrosis contiguous to occluded veins. The differences in the lesions may be explained by the difference in the duration and severity of a similar pathologic process.

The pathologic findings without the central nervous system consisted in Case 1 of advanced edema of the lungs, heart muscle, kidney, and pancreas. In Case 2, there were a few small foci of hemorrhagic infarction of the kidney associated with the signs of vasoparalysis of the small blood vessels, entirely similar to that described in the nervous system. In Case 3 there were vasothrombosis of the small vessels of the lungs and lobular pneumonia.

It is somewhat surprising how few are the histopathologic studies which concern this type of brain alteration. In the summer of 1923 Brown[3] at the Children's Hospital in Boston, observed six cases of rapidly fatal disease in children attended by symptoms referable to the central nervous system. In five of the cases, necropsy revealed intense edema and congestion of the brain. In the summer of 1924 Symmers[3] at Bellevue Hospital in New York, observed five cases presenting clinical and anatomical changes identical with those reported from Boston. Brown and Symmers termed their cases "acute serous encephalitis". No mention was made of the blood vessels in their reports. In spite of the fact that no inflammatory changes were observed the authors concluded that the disease belonged in the same general category with lethargic encephalitis.

We might assume that the cases described by Brown and Symmers are similar to those recorded here, but it is difficult to be sure because of the incompleteness of pathologic reports. Manifestly the clinical picture as well as the findings in the central nervous system do not warrant comparison with lethargic encephalitis.

Our studies indicate that we are dealing with brain damage secondary to functional vascular disturbances (sometimes termed "sludging"). The etiological factor of the circulatory disorder in these cases remains obscure. The disturbed water and electrolyte balance discussed by Doctor Weech might be causative, and should be considered in future studies.

Clinical Diagnosis

Encephalitis, type undetermined.

Doctor Weech's Diagnosis

Cerebral vascular disorder resulting from disturbance of electrolyte pattern in blood stream.

Anatomical Diagnosis

Functional vascular disturbance of small cerebral vessels (vasoparalysis, vasothrombosis).

Dr. Ferris: How do these changes differ from those described in such non-specific encephalopathies as we may see with various toxic states which also are associated with hypernea and changes in the level of consciousness?

Dr. Scheinker: Comparable changes in the nervous system have been noted in sulfa-

thiazole or carbon monoxide intoxication, arsenical encephalopathy, in trauma to the nervous system, and in a host of other conditions.

Dr. Weech: Because the skull which encloses the brain is a relatively rigid structure, even in infancy, cerebral edema is always a sharply limited process. But, edema fluid can accumulate in the extracellular tissue of brain at the expense of brain cells which become shrunken from dehydration. This was the type of lesion described by Yannet and Darrow as the result of artificially induced fever in young animals. I should therefore like to ask whether or not the pathological material from these cases was suitable for detecting possible changes in size of the glial and pyramidal cells?

Dr. Scheinker: I think one is able to measure cell size particularly with cellular stains or in the Bodian silver method. In cases of swelling due to trauma there appears to be swelling of nerve cells, glial cells, axis cylinders, and most myelin sheaths. We are able to rule out changes due to fixation.

Dr. Weech: Am I correct then in understanding that the brain cells in these cases showed no great change?

Dr. Scheinker: The nerve cells showed no advanced alterations. The earlier cellular change consisted of swelling. In later stages the edema was primarily extracellular.

Dr. George L. Engel: How often do you see respiratory alkalosis with fever when there is no other disturbance to account for the acid-base changes?

Dr. Weech: I should say that we are just beginning to recognize such disturbances. In the past the low CO_2 values were generally accepted as evidence of acidosis.

Dr. Rapoport: Ordinarily with fever in infants there is no respiratory alkalosis of any marked degree. As a matter of fact there usually is a mild acidosis.

Dr. Ferris: We studied adults with heat stroke, with temperatures from 106 to 114° F, and they all had extreme degrees of hyperventilation. The CO_2 content of the blood was as low as 30 vols.%. We weren't able to prove whether the low CO_2 was due to acidosis or alkalosis, because the pH was not measured, but I am convinced it was respiratory alkalosis.

There wasn't any marked disturbance of the CO_2 in these children. Therefore, a discussion of acid-base changes in relation to the brain pathology in these cases seems irrelevant.

Dr. Weech: It is possibly significant that after so much vomiting the CO_2 value was on the low side of normal rather than on the high side.

Dr. Josef Warkany: Is there any danger in treating these children with intravenous drips?

Dr. Charles D. Aring: The permeability of nervous system barriers is increased with alkalosis, hydration, hypoxia, or with inflammatory changes. Theoretically it seems that anything which might tend to decrease the effectiveness of the barriers of the central nervous system ought to be avoided.

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1946 LASKER AWARD

The Lasker Award, presented annually for outstanding service in the field of mental hygiene, will be given this year for the most significant experimental investigation into behavior deviation, it was announced by Dr. George S. Stevenson, Medical Director, The National Committee for Mental Hygiene, 1790 Broadway, New York 19, New York.

Nominations with supporting data, which will be presented to an anonymous jury chosen for its competence to judge accomplishment in the field selected, are now being accepted by the Committee. The work of the candidate must either have been accomplished or have been tested and won general acceptance, approximately within the past year.

Presentation of the $1000 award is made each fall at the annual meeting of the Committee, which will be held this year on October 30 and 31 at the Hotel Pennsylvania.

The Lasker Award was established in 1944 by the Albert and Mary Lasker Foundation, and was

presented in that year to Colonel William C. Menninger, M.C., Chief Consultant in Neuropsychiatry, Office of the Surgeon General, U. S. Army, for his outstanding contribution to the mental health of men and women of the U. S. Armed Forces.

In 1945 the award was conferred jointly on Dr. John Rawlings Rees, Consultant in Psychiatry to the Directorate of Psychiatry of the British Army, and Major General G. Brock Chisholm, Deputy Minister of National Health, Federal Department of National Health and Welfare, Canada, for outstanding service in rehabilitation.

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BOOK REVIEW

HYPNOANALYSIS. By Lewis R. Wolberg. New York: *Grune and Stratton, Inc.*, 1945. Pp. xviii-342.

An adequate review of this book is not possible within present space limitations, nor is one warranted since the book ought to be read in its entirety. It is a straightforward, easily readable account of the therapy of a deteriorated hospitalized schizophrenic patient by a combination of hypnosis and psychoanalysis. This is followed by a systematic, comprehensive, and readily understandable exposition of hypnotic techniques and psychoanalytic procedures. Neither special training nor experience is required to read this book with full, appreciative understanding of hypnosis and psychoanalysis as comprehensible and legitimate scientific procedures in the treatment of mental disorders. Also, the reader trained both in psychoanalysis and hypnosis will find a wealth of ideas and understandings which will serve to correct many current misunderstandings and actually to constitute a source of inspiration to enjoy both a knowledge of mental disease and a realization that genuine therapy is an actuality through an adequate recognition and utilization of relatively simple and ordinary psychological processes.

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